Belaich, J Sleep Disor: Treat Care 2020, 9:6
DOI: 10.37532/jsdtc.2020.9(6).249

Journal of Sleep Disorders: Treatment & Care

Research Article A SCITECHNOL JOURNAL

Brain Plasticity, Oxidative Stress and Sleep, What's the Link?

Rachida Belaich*

Abstract

A large number of existing studies have examined the role of sleep, since it seems to be a crucial need and involves both physiologic and behavioral processes. It is well established that sleep promotes various cognitive functions, participates in the consolidation of fresh memory traces, and has effects in brain maturation. However, sleep remains a scientific question, yet general consensus exists today that sleep is strictly essential for the creation of memory and long-term memory circuits, linked to learning and, in general, to the mechanisms of neural plasticity.

In the present paper we will first explore what findings have already been reported, linking sleep with brain plasticity. We will then review research associating sleep with oxidative processes. In closing, we will discuss what relation can linking sleep, brain plasticity and oxidative stress. From these facts one may conclude that sleep and brain plasticity are strongly related. The transition between wakefulness and sleep involves profound changes in motor control, cognition, brain activity, and consciousness. A working description of sleep as an electrophysiologically and behaviorally defined state has been well-known. Nevertheless, the function of sleep is not fully elucidated.

A better understanding of the changes of oxidative stress and sleep is crucial for developing methods that directly enhance sleep dependent plasticity, since promoting sleep may be useful to restore synaptic plasticity in different pathological conditions. Also, a better understanding of brain mechanisms controlling sleep/wake states would allow us to gain new insights on the functions of sleep and improve the capacity to treat sleep disorders.

Keywords

Sleep; Brain plasticity; Oxidative stress; Cognitive functions; Memory

Abbreviations

BDNF: Brain-Derived Neurotrophic Factor; DG: Dentate Gyrus; EEG: Electroencephalography; fMRI: Functional Magnetic Resonance Imaging; LTP: Long-Term potentiation; NREM: Non- Rapid Eye Movement; REM: Rapid Eye Movement; ROS: Reactive Oxygen Species; SHY: Synaptic Homeostasis Hypothesis; SWS: Slow Wave Sleep

Introduction

Sleep appears to serve an absolute vital function to most living organisms; it benefits both the body and the brain. Its known that Humans spend about one-third of their lives asleep. Despite its ubiquity, the fundamental functions of sleep remain elusive. Nevertheless, different hypotheses were suggested to explain it.

Sleep is recognized not simply as a resting state, but as an active state characterized by reduced alertness and responsiveness that is rapidly reversible. There are two main types of sleep: Rapid Eye Movement (REM) sleep, also known as paradoxical sleep, during which rapid, binocularly symmetrical eye movements occur. It is characterized by low-voltage fast Electroencephalography (EEG); it was discovered by Aserinsky and Kleitman in 1953 and the EEG recordings are remarkably similar to that of the awake state. The second type is Non-REM (NREM) sleep, which is characterized by a regular occurrence of local and global slow cortical oscillations, visible at the level of the EEG as slow waves, neurons in the cortex and thalamus are bistable. The bistability of thalamic and cortical neurons during NREM sleep impairs cortical information transmission and cortico–cortical effective connectivity NREM sleep is further divided into four Stages from the lightest Stage 1 to the deepest Stage 4. Together, NREM sleep stages 3 and 4 are often known as Slow Wave Sleep (SWS) [1].

Sleep is supposed to be regulated by two types of mechanisms; the two processes are known to be able to work independently. The first mechanism is the molecular mechanisms that control the timing of sleep, underlying circadian regulation, it governs many physiological processes including production of hormones. This mechanism has been well characterized. The second, the sleep homeostasis mechanisms, that control the duration of sleep, yet are less well defined; the homeostatic regulation of REM sleep is different from NREM sleep [2, 3,4]. Some authors have suggested that REM sleep's homeostatic regulation is primarily related to NREM sleep preceding REM sleep rather than preceding wakefulness [3,4]. The schematic version of the types of sleep and his mechanisms presented in (Figure 1).

A large number of existing studies have examined the role of sleep, since it seems to be a crucial need and involves both physiologic and behavioral processes [5]. It is well established that sleep promotes various cognitive functions, participates in the consolidation of fresh memory traces, and has effects in brain maturation [6]. Furthermore, sleep and sleep cycles are essential for the development of the neurosensory and motor systems in the fetes and neonate [7]. Also, a study has shown that homeostatic regulation of REM sleep plays a key role in neural plasticity and deficits in this process are implicated in the development of many neuropsychiatric disorders [8].

Sleep remains a scientific question, yet general consensus exists today that sleep is strictly essential for the creation of memory and long-term memory circuits, linked to learning and, in general, to the mechanisms of neural plasticity [9].

In exchange, sleep deprivation could be responsible for irreparable damage, which is correlated with a variety of diseases. Also, a substantial amount of research has been conducted to understand the effect of chronic sleep loss [10]. Per se, recent data has revealed that disordered sleep is among the most consistent symptoms and may contribute to the progression of a variety of diseases, many of which are also associated with stress as well as oxidative processes [11-14].

***Corresponding author:** Dr. Rachida Belaich, PhD, 1Department of Neuroscience, Faculty of science, University of Fez, 45 Road Ichbilia, Mimosa Kenitra 14050, Morocco, Tel: +212 669 896 567; E-mail: belaichrachida@gmail.com

Received: October 16, 2020 **Accepted:** December 28, 2020 **Published:** January 04, 2021

doi: 10.37532/jsdtc.2020.9(6).249

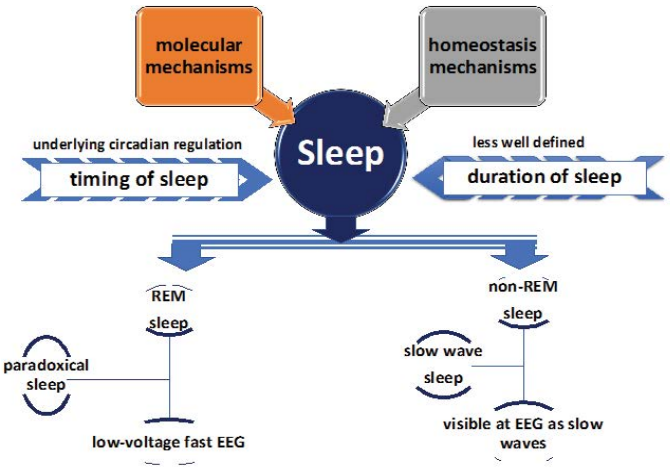

Figure 1: Schematic version of the two types of sleep: rapid eye movement (REM) sleep, characterized by low-voltage fast electroencephalography (EEG), non-REM (NREM) sleep or slow wave sleep, visible at the level of the EEG as slow waves. Sleep is regulated by two types of mechanisms: the molecular mechanisms that control the timing of sleep, underlying circadian regulation, and the sleep homeostasis mechanisms, that control the duration of sleep.

In the present paper we will first explore what findings have already been reported, linking sleep with brain plasticity. We will then review research associating sleep with oxidative processes. In closing, we will discuss what relation can be linking sleep and brain plasticity and oxidative stress.

Is Sleep Essential for Brain Plasticity?

" Every man can, if he so desires, become a sculptor of his own brain" [15]. This sentence is the first revelation of the existing of brain plasticity phenomena.

Human brain is capable for continuous functional changes over time, within certain boundaries. It is able to make new connections and change neural networks by both adding and pruning synapses [14]. In addition to plastic changes in brain function, both learning and development are associated with plastic changes in brain morphology [16]. The term 'neuroplasticity' was described for the first time by Jerzy Kanjorski, a Polish neuroscientist in 1948. Neuroplasticity include three essentials phenomena: synaptogenesis which is the inherent capacity of nerve tissue to form new interneuron connections or synapses; neurodegeneration that replace useless and non-functional neurons and neurogenesis by which new neurons are formed. Neuroplasticity shows that an adult brain can be adaptable, as a response to persistent intrinsic or extrinsic or to a lesion [17].

Two form of neuroplasticity are distinguished. The first one is structural neuroplasticity which refers to synaptic plasticity, it is a change in the strength between neurons (synapses), chemical or electric meeting points between brain cells. The synaptic plasticity is a process called Long-term Potentiation (LTP) that is believed to be a cellular mechanism of learning and memory. LTP is defined as a long-term enhancement of synaptic strength resulting from repeated activation of that synapse. The second one is functional neuroplasticity which depends upon two basic processes, learning and memory [16].

The sleep-wake cycle involves neurophysiological states like wakefulness, SWS and REM sleep. Different hypotheses were suggested to explain the functions of sleep, but there was consensus among the link with sleep, memory, learning and, in general, the mechanisms of neural plasticity. Memory formation depends on brain plasticity. If sleep is to be considered a critical mediator of memory consolidation, then evidence of sleep dependent plasticity would significantly support this claim.

The first line of evidence supporting a relationship between sleep and neural plasticity comes from that what is being accumulated during wake and reduced during sleep is actually not a molecule, but the overall synaptic strength of many neural circuits, which increases during wake because of learning and needs to be renormalized during sleep to avoid the high cost of plasticity from the increased need for energy and cellular supplies to the saturation of the ability to learn [18].

A causal relation between brain-derived neurotrophic factor (BDNF) expression during wakefulness and subsequent sleep homeostasis has been observed in vivo. BDNF plays an important role in a variety of neural processes during the development of both animals and humans. It has emerged as crucial mediator of neuronal plasticity [17,19].

Besides, some researchers have investigated the effects on sleep of a specific vasomotor task

that activates the right parietal cortex and seems to be related to a synaptic potentiation mechanism, which supports the hypothesis of the activation of a local homeostatic process during sleep. According to the synaptic homeostasis hypothesis sleep has a functional role in promoting the so-called synaptic downscaling [20].

Thus, scientific data suggests that sleep and the processes of brain plasticity are major players in time dependent processes of memory consolidation, helping integrate information for the long term. The purported mechanism by which sleep has such effect on memory is the reactivation and consolidation of recently potentiated synapses [21].

Indeed, studies suggest a close relationship between the electrical activity of NREM sleep and activation of glutamate receptors that contribute to learning, providing evidence for the role of NREM sleep in reactivation and maintenance of LTP. REM sleep is also implicated in LTP reprocessing [22].

At an electrophysiological level, which reflect collective changes in the brain, it reveals a profound link between sleep and neural

doi: 10.37532/jsdtc.2020.9(6).249

plasticity. Based on the synaptic homeostasis hypothesis (SHY) which proposes that the fundamental function of sleep is the restoration of synaptic homeostasis. In other word, when the brain goes off-line in sleep, renormalization of synaptic strength happens primarily [23,24].

Moreover, sleep seems to restore synaptic plasticity, with beneficial effects on learning processes since memory formation depends on brain plasticity. Several electrophysiological and imaging studies provide comprehensive support and mechanistic basis for the notion that sleep promotes cognitive processing [25].

Using brain imaging, authors have shown that patterns of brain activity expressed during training on a serial reaction time motor task reappear during subsequent REM sleep [26], Functional Magnetic Resonance Imaging (fMRI) has also been used to compare patterns of brain activation before and after a night of sleep. Results suggest that sleep-dependent motor learning is associated with a large-scale plastic reorganization of memory throughout several brain regions, allowing skilled motor movements to be executed more quickly, more accurately, and more automatically following sleep. Following a night of sleep, and relative to an equivalent intervening period awake, increased activation was identified in motor control structures of the right primary motor cortex [27,28].

Furthermore, the sleep-dependent visual texture discrimination task was used and test following sleep was associated with significantly better activation in an area of primary visual cortex corresponding to the visual target location [29].

In total, these results lead to the conclusion that sleep and brain plasticity seem to be strongly related which is schematized in (Figure 2).

Sleep disturbances and brain plasticity

Sleep deprivation is a common feature in our modern society, it may be acute and have been characterized in terms of one continuous extended wake episode, consisting of abnormalities in alertness/sleepiness, continuity or efficiency, duration and timing. The most common sleep disorders are: insomnia, dyssomnias, parasomnias, sleep disorders associated with mental, neurologic, or other medical disorders and proposed sleep disorders [30].

A substantial amount of research on human has been conducted to understand the former but more recently the effect of chronic sleep loss has also been investigated. Various types of sleep deprivation have been used. Studies indicate that REM sleep deprivation was particularly harmful for memory consolidation. Memory consolidation is impaired also in different clinical samples characterized by disturbed sleep. Patients with primary insomnia showed a decreased sleep-dependent memory consolidation in procedural and declarative learning associated with the reduction of REM sleep. Besides, sleep deprivation is usually followed by enhanced vulnerability to stress which can decrease BDNF production which can disturb neuronal plasticity. Thus, sleep deprivation may impair memory consolidation in part by reducing the synthesis of proteins needed to support synaptic plasticity [31,32].

Studies in humans have shown that sleep deprivation affects cortical systems involved in a variety of functions, including executive attention, working memory, and higher cognitive abilities. Additionally, electrophysiological studies were conducted to determine the effect of sleep deprivation on various forms of hippocampal synaptic plasticity. One prominent form of hippocampal synaptic plasticity is LTP. Sleep deprivation, which is known to impair learning, has been shown to negatively alter protein cascades necessary for LTP and attenuates it [33,34]. The (Figure 3) resume the effect of sleep deprivation on LTP and especially the influence of reduction of REM sleep on memory consolidation and synaptic plasticity. Together, these results suggest that sleep and sleep deprivation bi-directionally can impact the molecular signalling pathways that regulate synaptic strength and control brain plasticity, supporting the hypothesis of a direct relation between cortical plasticity and sleep regulation (Figure 4).

Another hypothesis suggests that comes from patients suffering from insomnia. These patients show decreased sleep-dependent memory consolidation, which is commonly considered an indicator for neural plasticity, in procedural and declarative learning. Results from human studies are consistent with the findings in animals, showing a sleep-dependent re-activation of brain regions involved in previous learning [35,36].

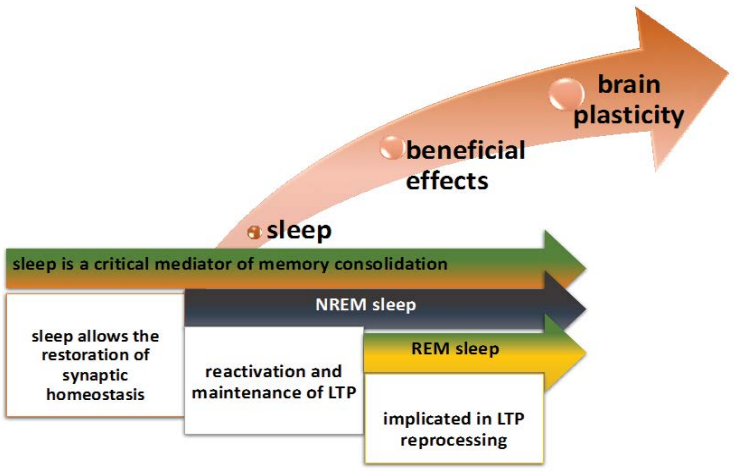

Figure 2: Sleep and brain plasticity seem to be strongly related which is schematized in this figure, sleep seems to restore synaptic plasticity, with beneficial effects on learning processes.

doi: 10.37532/jsdtc.2020.9(6).249

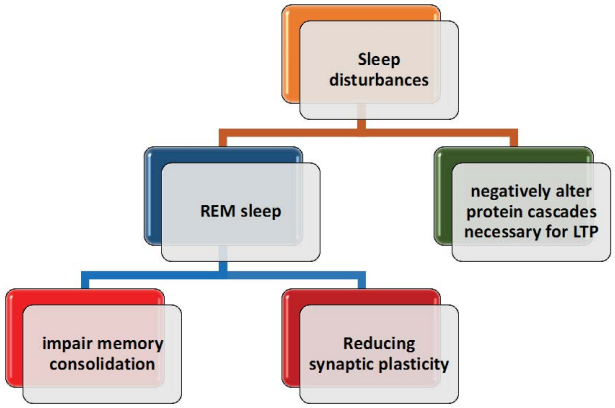

Figure 3: Resume the effect of sleep deprivation on LTP and especially the influence of reduction of REM sleep on memory consolidation and synaptic plasticity.

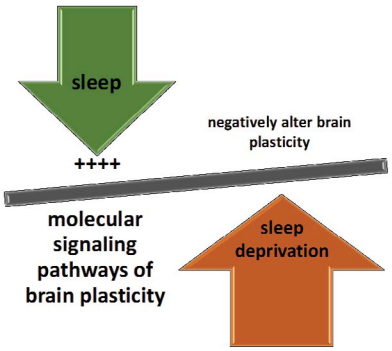

Figure 4: Sleep and sleep deprivation bi-directionally can impact the molecular signaling pathways that regulate synaptic strength and control brain plasticity, supporting the hypothesis of a direct relation between cortical plasticity and sleep regulation.

Oxidative Stress and Sleep

Sleep has important functions for every organ in the body, it is a restorative process that plays an important role in the balance of psychological and physical health.

Sleep disorders is correlated with a variety of diseases and have been implicated as a predictor for Alzheimer, Parkinson, and Huntington's diseases, many of these diseases are also associated with oxidative damage [37,38].

Here we show the evidence regarding the effects of sleep and the production of ant oxidative markers as protective elements.

Oxidative stress is defined as an imbalance between the production of free radicals which are Reactive Oxygen Species (ROS) and antioxidants defences. ROS are produced from molecular oxygen as a result of normal cellular metabolism. Higher production of ROS in body may cause damaging covalent modifications that change DNA structure, inhibit the function of proteins, lipids and can lead to cell death; neuronal accumulation of ROS is a plausible contributing factor in the pathogenesis of neurodegenerative diseases. The human body is equipped with a variety of antioxidants that serve to counterbalance the effect of oxidants [39].

The existing literature claimed, for instance, that sleep may let to decrease oxidative stress, this is supported by the fact that sleep can allow clearance of ROS accumulated in the brain during the wake state. The ROS and other oxidative stress markers could be accumulated in the brain tissue during wakefulness, and after reaching out a threshold, they could behave as sleep promoters. Therefore, sleep represents a state with an increased antioxidant activity which promotes a brain protection against free radicals via a diminution in oxidant production. Thus, it seems that sleep induces repair, restoration and detoxification. Besides, it has been proposed that, during sleep, uridine and glutathione may facilitate the oxidative detoxification of the brain by potentiating GABAergic transmission and inhibiting glutamatergic transmission (Figure 5). Furthermore, sleep represents a state with an increased antioxidant activity which promotes a brain protection against free radicals via a diminution in oxidant production [40].

Several theories about the functions of sleep repose on the assumption that wakefulness represents an oxidative challenge for the brain. Wakefulness involves high neuronal metabolism to maintain neuronal electrical potentials, which requires a great amount of oxygen, resulting in a significant production of oxidants. It is also suggested that sleep deprivation compromises the immune function in humans by provoking a pro-inflammatory response via increased cytokine secretion. Additionally, there is evidence indicating that enhanced pro-inflammatory cytokine signalling may promote ROS generation and lead to oxidative damage which is schematized in (Figure 6).

Consistent with these hypotheses, sleep deprivation could be responsible for recruiting neurobiological mechanism related with stress as well as oxidative processes [41].

doi: 10.37532/jsdtc.2020.9(6).249

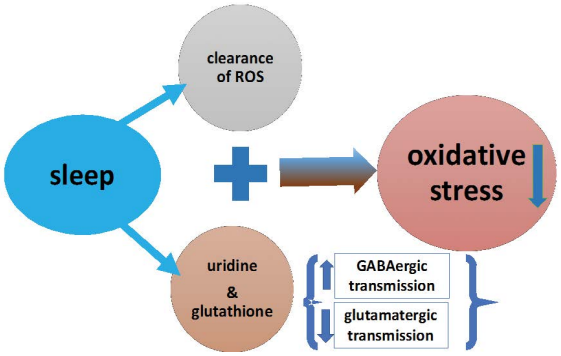

Figure 5: Sleep can allow clearance of ROS accumulated in the brain during the wake state. Besides, uridine and glutathione may facilitate the oxidative detoxification of the brain by potentiating GABAergic transmission and inhibiting glutamatergic transmission.

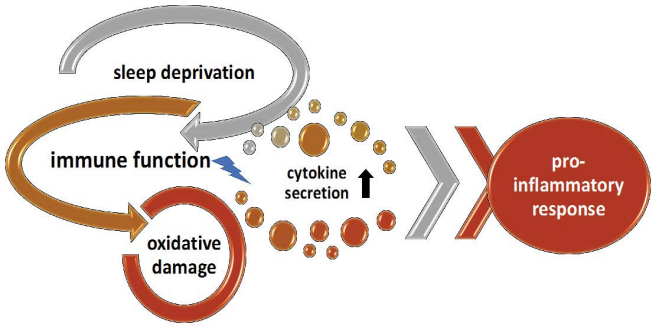

Figure 6: Sleep deprivation compromises the immune function by provoking a pro-inflammatory response via increased cytokine secretion which may lead to oxidative damage.

Oxidative Stress and Brain Plasticity

The brain is a highly plastic organ that undergoes constant changes in structure and function in response to experience. Also, this organ has a very active oxidative metabolism compared with other organs because of its rich lipid content, high energy demand, and weak antioxidant capacity. This makes brain particularly vulnerable to oxidative stress [42].

Several reports indicate that ROS play key roles in hippocampal LTP formation,

but high ROS concentrations reportedly diminish LTP and synaptic signalling and brain plasticity mechanisms, which can cause significant neuronal damage. Therefore, it seems highly plausible that oxidative stress in the brain compromises bio-chemical integrity of the hippocampus and the amygdala. It is well known that the hippocampal Dentate Gyrus (DG) system regulates structural plasticity, besides it is becoming increasingly apparent that ROS increase plays key roles in the functional and structural changes that mediate hippocampal synaptic plasticity and hippocampus-dependent memory formation. Thus, oxidative damage of DG function may impair structural plasticity and disrupt synaptic neurotransmission [43].

Further, some data suggest that oxidative stress can interact with BDNF to modulate synaptic plasticity and cognitive function. These data present evidence that oxidative stress could be an important factor stimulating BDNF expression. BDNF is a potent neurotrophic found in many tissues, including hippocampus. This peptide stimulates neuroplasticity, through direct mediation of the formation of new neuronal circuits, neuronal survival and synaptogenesis [44].

Several studies showed the implications of oxidative stress in the brain plasticity and there is much literature to demonstrate that oxidative stress underlies the age-dependent decline of synaptic plasticity mechanisms required for cognitive functions [45-48].

The precise chain of events occurring within the central nervous system that potentially causes or leads to the implications of oxidative stress in the brain plasticity is an interesting topic. Since ROS may regulate neuronal activity and elicit negative effects at the same time, the distinction between beneficial and deleterious consequences is unclear [49,50].

Conclusion

From these facts one may conclude that sleep and brain plasticity are strongly related. The transition between wakefulness and sleep involves profound changes in motor control, cognition, brain activity, and consciousness. A working description of sleep as an electro physiologically and behaviourally defined state has been well-known. Nevertheless, the function of sleep is not fully elucidated.

A better understanding of the changes of oxidative stress and sleep is crucial for developing methods that directly enhance sleep dependent plasticity, since promoting sleep may be useful to restore synaptic plasticity in different pathological conditions. Also, a better understanding of brain mechanisms controlling sleep/wake states would allow us to gain new insights on the functions of sleep and improve the capacity to treat sleep disorders

Conflict of Interest

The author declares that he has no conflict of interest.

Ethical Approval

This article does not contain any studies with human participants or animals performed. Ethical approval statement is therefore not applicable.

Informed Consent

For this type of study formal consent is not required. Informed consent statement is therefore not applicable.

Reference

1. Aserinsky E, Kleitman N (1953) Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. Science 118: 273–274.
2. Alada R, Cirelli C, Sehgal A (2017) Molecular mechanisms of sleep homeostasis in flies and mammals. Cold Spring Harb Perspect Biol 9: a027730.
3. Dijk DJ, Landolt HP (2019) Sleep Physiology, Circadian Rhythms, Waking Performance and the Development of Sleep-Wake Therapeutics. Handb Exp Pharmacol 253:441-481.
4. Vyazovskiy VV, Delogu A (2014) NREM and REM Sleep: Complementary Roles in Recovery after Wakefulness. Neuroscientist 20: 203-219.
5. Frank MG, Heller HC (2019) The function(s) of sleep. Handb Exp Pharmacol.
6. Ednick M, Cohen AP, McPhail GL, Beebe D, Simakajornboon N et al. (2009) A review of the effects of sleep during the first year of life on cognitive, psychomotor,and temperament development. Sleep 32: 1449–1458.
7. Tarullo AR, Balsam PD, Fifer WP (2011) Sleep and Infant Learning. Infant Child Dev 20:35-46.
8. Datta S, Knapp CM, Koul-Tiwari R, Barnes A (2015) The homeostatic regulation of REM sleep: A role for localized expression of brain-derived neurotrophic factor in the brainstem. Behav Brain Res 292: 381-392.
9. Rasch B, Born J (2013) About sleep's role in memory. Physiol Rev 93: 681-766.
10. Alkadhi K, Zagaar M, Alhaider I, Salim S, Aleisa A (2013) Neurobiological consequences of sleep deprivation. Curr neuropharmacol 11: 231–249.
11. McCoy JG, Strecker RE (2011) The cognitive cost of sleep lost. Neurobiol Learnand Mem 96: 564–582.
12. Datta S, Maclean RR (2007) Neurobiological mechanisms for the regulation of mammalian sleep-wake behavior: reinterpretation of historical evidence and inclusion of contemporary cellular and molecular evidence. Neurosci Biobehav Rev 31: 775–824.
13. Xu L, Jiang CQ, Lam TH, Liu B, Jin YL et al. (2011) Short or Long Sleep Duration Is Associated with Memory Impairment in Older Chinese: the Guangzhou Biobank Cohort Study. Sleep 34:575-580.
14. Lima AM, de Bruin VM, Rios ER, de Bruin PF (2014) Differential effects of paradoxical sleep deprivation on memory and oxidative stress. Naunyn Schmiedebergs Arch Pharmacol 387: 399-406.
15. Demarin V, Bedeković MR, Puretić MB, & Pašić MB (2016) Arts, brain and cognition. Psychiatr Danub 28: 343–348
16. Kulak W, Sobaniec W (2004) Molecular mechanisms of brain plasticity: neurophysiologic and neuroimaging studies in the developing patients. Rocz Akad Med Bialymst 49:227–236.
17. Cajal SRY(1955) Histologie du système nerveux de l'homme & des vertébrés. Consejo Superior de Investigaciones Científicas, Instituto Ramon y Cajal.
18. Mateos-Aparicio P, Rodríguez-Moreno A (2019) The Impact of Studying Brain Plasticity. Front Cell Neurosci 13: 66.
19. Nissen C, Kloepfer C, Feige B, Piosczyk H, Spiegelhalder K et al. (2011) Sleep-related memory consolidation in primary insomnia. J Sleep Res 20: 129-136.
20. Sangiovanni E, Brivio P, Dell'Agli M, Calabrese F (2017) Botanicals as Modulators of Neuroplasticity: Focus on BDNF. Neural Plast 5965371.
21. Puentes-Mestril C, Aton SJ (2017) Linking Network Activity to Synaptic Plasticity during Sleep: Hypotheses and Recent Data. Front Neural Circuits 11: 61.
22. Peigneux P, Smith C (2010) Memory processing in relation to sleep. In: Kryger M, Roth T, Dement W (eds) Principles and practice of sleep medicine, 17th edn. Elsevier Philadelphia 335–347.
23. Abel T, Havekes R, Saletin JM, Walker MP (2013) Sleep, plasticity and memory from molecules to whole-brain networks. Curr Biol 23: R774-R788.
24. Zhang MQ, Li R, Wang YQ, Huang ZL (2017) Neural Plasticity Is Involved in Physiological Sleep, Depressive Sleep Disturbances, and Antidepressant Treatments. Neural Plast 5870735
25. Tononi G, Cirelli C (2014) Sleep and the price of plasticity: from synaptic and cellular homeostasis to memory consolidation and integration. Neuron 81: 12–34.
26. Censor N, Sagi D, Cohen LG (2012) Common mechanisms of human perceptual and motor learning. Nat Rev Neurosci 13:658–664.
27. Rasch B, Born J (2013) About sleep's role in memory. Physiol Rev 93: 681–766.
28. Walker MP (2009) The role of slow wave sleep in memory processing. Journal of clinical sleep medicine : JCSM : official publication of the American Academy of Sleep Medicine, 5: S20–S26
29. Dayan E, Cohen LG (2011) Neuroplasticity subserving motor skill learning. Neuron 72: 443–454.
30. Ekman M, Kok P, de Lange FP (2017) Time-compressed preplay of anticipated events in human primary visual cortex. Nat Commun 8: 15276
31. Pavlova MK, Latreille V (2019) Sleep Disorders American Journal of Medicine 132: 292-299.
32. Havekes R, Vecsey CG, Abel T (2012) The impact of sleep deprivation on neuronal and glial signaling pathways important for memory and synaptic plasticity. Cell Signal 24:1251-1260.
33. Vgontzas AN, Fernandez-Mendoza J, Liao D, Bixler EO (2013) Insomnia with Objective Short Sleep Duration: the Most Biologically Severe Phenotype of the Disorder. Sleep Med Rev 17: 241-254.
34. Havekes R, Vecsey CG, Abel T (2012) The impact of sleep deprivation on neuronal and glial signaling pathways important for memory and synaptic plasticity. Cell Signal 24: 1251-1260.
35. Gorgoni M, D'Atri A, Lauri G, Rossini P, Ferlazzo F et al. (2013) Is Sleep Essential for Neural Plasticity in Humans, and How Does It Affect Motor and Cognitive Recovery? Neural plast 103949.
36. Tononi G, Cirelli C. 2014. Sleep and the price of plasticity: From synaptic and cellular homeostasis to memory consolidation and integration. Neuron 81: 12–34.
37. Prince TM, Abel T(2013) The impact of sleep loss on hippocampal function. Learn Mem 20: 558–569.
38. Sterniczuk R, Theou O, Rusak B, Rockwood K (2013) Sleep Disturbance is Associated with Incident Dementia and Mortality. Curr Alzheimer Res 10: 767-775
39. Lázár A, Panin F, Goodman A, Lazic S, Lazar Z et al. (2015) Sleep, But No Metabolic, Deficits In Pre-Manifest Huntington's Disease. Ann Neurol 78: 630-648.
40. Deveci G, Tek N (2019) A Mini Review of Astaxhantin and Oxidative Stress in Aging.
41. Yeh Ting-T, Hsieh YW, Wu Ching y, Wang JS, Lin KC et al.(2017) A Preliminary Investigation of the Association of Sleep With Inflammation and Oxidative Stress Biomarkers and Functional Outcomes After Stroke Rehabilitation. Sci Rep 7: 8634.
42. Alkadhi K, Zagaar M, Alhaider I, Salim S, Aleisa A (2013) Neurobiological Consequences of Sleep Deprivation. Curr Neuropharmacol 11: 231-249.
43. Salim S (2017) Oxidative Stress and the Central Nervous System. J Pharmacol Exp Ther 360: 201–205.
44. Hidalgo C, Arias-Cavieres A (2016) Calcium, Reactive Oxygen Species, and Synaptic Plasticity. Physiology 31: 201-215.
45. Freitas DA, Rocha-Vieira E, Soares BA, Nonato LF, Fonseca SR et al. (2018) High-intensity interval training modulates hippocampal oxidative stress, BDNF and inflammatory mediators in rats. *Physiol Behav* 184: 6–11.
46. De Pasquale R, Beckhauser TF, Francis-Oliveira J (2016) Reactive Oxygen Species: Physiological and Physiopathological Effects on Synaptic Plasticity. J Exp Neurosci 10: 23-48.

doi: 10.37532/jsdtc.2020.9(6).249

47. Belaich R, Boujraf S, Housni A, Maaroufi M, Batta F (2014). Assessment of Hemodialysis Impact by Polysulfone Membrane on Brain Plasticity Using BOLD-fMRI. Neuroscience 288: 94-104

48. Boujraf S, Belaich R, Housni A, Maaroufi M, Tizniti S (2017) Blood Oxygenation Level-Dependent Functional MRI of Early Evidences of Brain Plasticity after Hemodialysis Session by Helixone Membrane of Patients with Indices of Adrenal Deficiency. Ann Neurosci 24: 82-89.

49. Belaich R, Boujraf S, Benzagmout M, Magoul R, Maaroufi M et al. (2016) Implications of oxidative stress in the brain plasticity originated by fasting: a BOLD-fMRI study. Nutr Neurosci 20: 1-8.

50. Belaich R (2019) Imagerie par résonance magnétique fonctionnelle cérébrale : concept BOLD et compréhension théorique du vieillissement neurocognitif. NPG Neurologie - Psychiatrie - Gériatrie 19: 129-136

Author Affiliations Top

Department of Neuroscience, Faculty of science, University of Fez, Morocco